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**Endogenous Hormonal and Growth Factor
Responses to Heavy Resistance Exercise Protocols**

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Running Head: Hormonal Responses to Resistance Exercise

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ABSTRACT

To examine endogenous anabolic hormone and growth factor responses to various heavy resistance exercise protocols (HREPs), nine male subjects performed each of six randomly assigned HREPs which consisted of identically ordered exercises carefully designed to control for load (5RM vs 10RM), rest period length (1 min vs 3 min) and total work (J) effects. Serum human growth hormone (hGH), testosterone (T), somatomedin-C (SM-C), serum glucose and whole blood lactate (HLA) concentrations were determined pre-exercise, mid-exercise (i.e after 4 of 8 exercises), and at 0, 5, 15, 20, 60, 90 and 120 min post-exercise. All HREPs produced significant ($p < 0.05$) increases in serum T concentrations although the magnitude and frequency above resting values varied across HREPs. The highest hGH concentrations were observed consequent to high total work, 1 min rest periods and 10RM load. All HREPs did not produce increases in serum hGH. The pattern of SM-C increases varied among HREPs and did not follow hGH changes. These data suggest that the release patterns and the magnitude of increases are functions of the type of HREPs utilized. Thus, all HREPs may not effect muscle and connective tissue growth in the same manner due to differences in hormonal and growth factor responses.

Key Words: human growth hormone, somatomedins, insulin-like growth factors, testosterone, resistance exercise, lactate, males.

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In vivo and in vitro investigations have demonstrated that several hormones (e.g. growth hormone, testosterone) and growth factors (e.g. somatomedins) are involved with skeletal muscle tissue growth and development (10). Yet, the exact mechanism(s) by which these growth promoting actions occur remain unclear. Heavy resistance training has been shown to be a potent stimulus for muscle cell hypertrophy (25,30). This may be due, in part, to exercise-induced increases in endogenous anabolic hormones and growth factors.

To date, studies have generally indicated that acute heavy resistance exercise stimulates an increase in peripheral blood concentrations of testosterone (T) in males (8,12,18,28,33). Furthermore it has been suggested that training may influence resting values of testosterone (13,14,15). Limited data also suggests that human growth hormone (hGH) may increase in response to an acute bout of resistance exercise (24,28,32). Van Helder et al. (32) have demonstrated that hGH elevations may be dependent upon specific exercise characteristics such as the load utilized and frequency of lifting exercise. To our knowledge, no data exist regarding somatomedin-C (SM-C) responses to heavy resistance exercise protocols.

Few attempts have been made to examine the specific influence of program design variables (e.g. load, rest period length), within a total heavy resistance exercise protocol (HREP), on hormonal response patterns (18). Thus, the purpose of this investigation was to determine the impact of load, rest period length and total work on serum T, hGH and SM-C response patterns during and following different heavy resistance exercise protocols HREPs. These data should help provide insights to mechanisms which may mediate exercise-induced tissue growth consequent to heavy resistance training, and provide a basis for further investigations examining the link between neuroendocrine responses and tissue growth.

METHODS

Nine healthy male subjects gave informed written consent to participate in this investigation. The physical characteristics of the subjects were ($\bar{x} \pm 1$ SD): age 24.66 ± 4.27 (yrs), height 178.41 ± 7.77 (cm), body mass 81.08 ± 12.03 (kg), maximal oxygen consumption ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) 54.17 ± 4.63 , body fat 15.96 ± 4.18 (%). All subjects had recreational experience with resistance training but none were competitive lifters. None of the subjects had a medical history of any endocrine disorder and none were on medications or hormonal therapy. Furthermore, subjects reported no history of anabolic steroid use. Urine testing of all subjects verified those reports during the study.

A minimum of two weeks were utilized for experimental protocol familiarization, descriptive testing and load verifications for each experimental exercise protocol. Percent body fat was determined using hydrostatic weighing with a computer interfaced load cell and standard body composition methodology previously described (9,35). Maximal oxygen consumption ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was determined utilizing a continuous treadmill protocol and plateau criteria previously described (4).

Each HREP was performed in random order. Subsequent statistical analysis demonstrated no order effects. The experimental design of each protocol is shown in Table 1. Six different HREPs were used in this investigation. Of the six protocols there were two primary workouts, weight training protocol-1 (WTP-1) and weight training protocol-2 (WTP-2). The WTP-1 workout was a five repetition maximum (5 RM) based workout which incorporated longer rest intervals (i.e. three minutes) and heavier weight (5 RM) lifted. The WTP-2 was a 10 RM based workout with one minute rest between sets. The WTP-1 protocol is typically utilized for "strength" training, while the WTP-2 protocol is

typically used by bodybuilders for increases in muscular hypertrophy (19). Additional workout variations of these primary workouts were performed in order to help determine if differences in the effects of the two types of exercise protocols were associated with the load lifted, rest period length or total work. The same exercise order was used in all of the exercise protocols. Thus, two exercise series were utilized in this study with each series having a primary workout (WTP-1 or WTP-2), and a load control and rest control that were matched for total work (J) of the primary workout. Series 1 (i.e. WTP-1, load and rest controls) had a lower ($p < 0.05$) total work ($49,161 \pm 10,100$ vs $59,859 \pm 12,675$ J) compared to Series 2 (i.e. WTP-2, load and rest controls). In Series 1, the load control was the same as WTP-1 (i.e. 3 min rest, same total work), however, subjects used a lighter weight (10 RM), so that they could perform ten instead of five repetitions per set. The rest control was identical to WTP-1 except one min rest periods were utilized instead of three min. In Series 2, the variations followed the same pattern (i.e. varying the load and rest period length). This design allowed for a more quantitative approach to examine responses to heavy resistance exercise due to specific changes in the exercise protocols.

All exercises were structured proportionally for each subject with grip widths and positions marked and kept constant for each exercise. The matching of the total work between workouts was performed by a computer program which, given a specific exercise, weight and number of repetitions, calculated the number of repetitions required to produce the same total work using a different weight. Lifting work was calculated as weight times vertical distance moved per repetition times number of repetitions. The program took into consideration the vertical distance moved of both the iron plates and the centers of gravity of the lifters body segments. These distances were

obtained from measurements on the subjects and equipment when they were in the starting and ending exercise positions. Anthropometric tables were used to locate body segment centers of gravity and estimate body segment weights from total body weight (36).

Experimental Protocol. One week separated each randomized experimental protocol. Prior to each test, subjects refrained from ingestion of alcohol or caffeine for 24 hrs prior to testing. Subjects did not perform any strenuous exercise for 48 hrs prior to the experimental exercise session. Dietary analysis (Nutri-Cal, PCD System Inc., Penn Yan, NY) for the 3 days prior to each experimental session demonstrated normal RDA caloric, nutrient, vitamin and mineral intakes. Prior urine nitrogen determinations verified all subjects to be in the normal range for positive nitrogen balance prior to each test session.

Subjects reported for the experimental session and venous blood samples were obtained in a slightly reclined seated position which was used for all samples. For each subject all testing was conducted at the same time of day (0800 hr) to reduce the effects of any diurnal variations on the hormonal concentrations. Prior to obtaining a resting blood sample, a 20 min equilibration period was utilized. Subjects knew they would not immediately start to exercise after the resting blood sample was obtained. The exercise protocol started 10 min after the resting blood sample was drawn. This procedure was shown during pilot testing to eliminate any significant anticipatory increases in hormonal responses previously thought to effect the examination of exercise responses (18). Water intake was allowed ad libitum throughout the exercise protocols and recovery. The venous blood samples were obtained from an indwelling cannula in a superficial arm vein kept patent with isotonic saline ($30 \text{ mL} \cdot \text{hr}^{-1}$). Blood samples were obtained pre-exercise, mid-

exercise (i.e. after 4 exercises) and at 0 (immediate-post), 5,15,30,60,90 and 120 min following each exercise protocol. All blood samples were processed and stored at -120°C until analyzed.

Biochemical Analyses. Whole blood lactate and serum glucose concentrations were determined in duplicate via a Lactate Analyzer-640 (Wolverine Med Inc., Grand Rapids, MI) and a 23-Glucose Analyzer (Yellow Springs, Inc., Yellow Springs, OH). Hemoglobin was analyzed in triplicate using cyanmethemoglobin method (Sigma Chemical Co., St. Louis, MO) and hematocrit was analyzed in triplicate utilizing standard micro-capillary technique. The percent changes in plasma volume were calculated according to equations by Dill and Costill (6).

Serum, testosterone, human growth hormone and somatomedin-C concentrations were determined utilizing radioimmunoassay procedures. All samples (run in duplicate) were decoded only after analyses were completed (i.e. blinded analysis procedure). Determinations of different serum immunoreactivity values were accomplished with the use of a Beckman 5500 gamma counter and on-line data reduction system. Serum samples were analyzed in duplicate for testosterone using an ^{125}I solid phase radioimmunoassay (Diagnostic Products Corp., Los Angeles, CA) was sensitive to detection limit of 0.38 nmol/L. Intra- and inter-assay variances were calculated to be less than 3.2% and 4.9% respectively. Serum samples were analyzed in duplicate for human growth hormone was measured utilizing an ^{125}I liquid phase radioimmunoassay with double antibody technique (Cambridge Medical Diagnostics, Billerica, MA) was sensitive to a detection limit of 0.24 ug/L. Intra- and inter-assay variances were calculated to be less than 4.4% and 4.9% respectively. Serum samples were analyzed in duplicate for somatomedin-C (insulin-like growth factor I) was measured using using an ^{125}I double antibody disequilibrium

radioimmunoassay with a preliminary ODS-silica extraction procedure (IncStar Corp., Stillwater, MN) was sensitive to a detection limit of < 2.0 nmol/L. Intra- and inter-assay variances were less than 4.6% and 4.8% respectively.

Statistical analyses of the data were accomplished utilizing analysis of variance with repeated measures. Post-hoc pairwise differences were determined using a Tukey test. Pearson product-moment correlation coefficients were performed on the data set. Significance in this study was chosen at $p < 0.05$.

RESULTS

Figure 1 shows the responses of serum testosterone to the various HREPs. In Series 1, WTP-1 significantly elevated testosterone (T) values, above rest at mid-exercise and at 0,5,15 and 90 min post-exercise. When the load was changed to 10RM (load control), but total work and rest kept constant, serum T values significantly increased above rest at 5 and 15 min post-exercise. Conversely, when only the rest period length was reduced to 1 min (rest control), significant increases in serum T were observed at mid-exercise and at 0,5,15 and 90 min following the exercise protocols. In addition, WRP-1 values were significantly higher than the load and rest controls at 30 min post-exercise and the load control at 60 min post-exercise.

In Series 2, WTP-2 significantly elevated serum T values above resting levels at mid-exercise and at 0,5 and 15 min post-exercise (Figure 1). When the load was increased to 5 RM (load control), but total work and rest kept constant, significant increases above rest were again observed at mid-exercise, and at 0,5 and 15 min post-exercise. When the rest period was increased to 3 min (rest control) significant elevations in serum T were

observed at mid-exercise and 120 min post-exercise. WTP-2 and the load control protocols had significantly higher serum T values at mid-exercise and at 0,5 and 15 min post-exercise than the rest control. WTP-2 and the rest control were also significantly different at 120 min post-exercise (WTP-2 < rest control).

WTP-1 load control serum T were significantly lower than WTP-2 and WTP-2 load control values at 0 min post-exercise. Furthermore, WTP-2 rest control values were significantly lower than WTP-1 and WTP-1 rest control at 0 and 5 min post-exercise. WTP-2, WTP-2 rest control, WTP-2 load control were significantly less than WTP-1 and WTP-1 rest control at 90 min post-exercise.

Figure 2 shows the responses of human growth hormone (hGH) to the various HREPs. In Series 1, WTP-1 significantly elevated serum hGH concentrations above rest at mid-exercise and at 0 min post-exercise. When the load was changed to 10RM keeping the total work and rest constant no significant increases in serum hGH were observed. When the rest was decreased to 1 min, significant increases in serum hGH concentrations above resting values were observed at 0 and 5 min post-exercise.

In Series 2, WTP significantly elevated serum hGH concentrations at mid-exercise and at 0,5,15 and 30 min post-exercise (Figure 2). When the load was increased (5RM) or the rest increased (3 min) no significant increases above resting concentration were observed.

Figure 3 shows the responses of serum somatomedin-C (SM-C) to the various HREP. In Series 1, WTP-1 significantly elevated serum SM-C concentrations above rest at 0 and 5 min post-exercise. When the load was decreased to 10RM with rest and total work kept constant, significant increases in serum SM-C were observed above rest at 90 min post-exercise. When rest was reduced to 1

min, significant increases in serum SM-C above resting concentrations were observed at 30 min post-exercise.

In Series 2, WRP-2 resulted in a significant increase in serum SM-C at 90 min post-exercise (Figure 3). When the load was increased to 5RM significant increases in serum SM-C were observed at mid-exercise. When the rest period was increased to 3 min, significant increases in serum SM-C were observed 5 min post-exercise.

In Series 1, the load control (90 min post-exercise) and rest control (30 min post-exercise) HREP responses of serum SM-C values were greater than values for Series 2 HREP at those respective timepoints (Figure 3). The rest control in Series 2 at 5 min post-exercise had values significantly higher than Series 1 HREP at that timepoint.

Figure 4 shows the responses of serum glucose (panel A) and whole blood lactate (panel B), respectively. No HREP in Series 1 or 2 elicited any significant changes in serum glucose concentrations. Whole blood lactate (HLA) (panel B) in WTP-1 significantly elevated HLa concentrations at mid-exercise and at 0,5,15 min post-exercise. Changing the load to a 10RM while keeping the total work and rest periods constant resulted in no significant increases in HLa concentrations. When the rest periods were reduced to 1 min, significant increases in HLa occurred at mid-exercise, and at 0,5 and 15 min post-exercise. These rest control values were significantly higher than WTP-1 and load control values at mid-exercise, 0 and 5 min post-exercise.

In Series 2, WTP-2 significantly elevated HLa above resting concentrations at mid-exercise, and at 0,5,15,30 and 60 min post-exercise. Both the load and rest controls in Series 2 demonstrated increases in HLa at mid-exercise, and at 0,5 and 15 min post-exercise. WTP-2 HLa values at mid-exercise, and at

0,5,15,30 and 60 min post-exercise were significantly greater than load and rest HREP HLa values at these timepoints.

WTP-2 HLa concentrations at mid-exercise, and at 0,5,15 and 30 min post-exercise were significantly higher than Series 1 WTP-1 and load control at those timepoints. Series 2 load and rest controls HLa values were also greater than Series 1 WTP-1 and load control HLa values at mid-exercise and at 0,5 and 15 min post-exercise.

The hormonal responses were corrected for % changes in plasma volume so as to reduce the influence of plasma volume shifts during the different exercise protocols on serum concentrations. Changes in plasma volume shifts during recovery were negligible. The greatest % change in plasma volume were observed pre- to post-exercise and were as follows ($\bar{X} \pm 1$ SD): Series 1: WTP-1 = $-7.68 \pm 4.28\%$, load control = $-3.58 \pm 4.25\%$ and rest control = $-4.86 \pm 2.54\%$; Series 2: WTP-2 = $-8.22 \pm 5.52\%$, load control = $-3.37 \pm 2.74\%$ and rest control = $-2.77 \pm 2.31\%$.

DISCUSSION

Heavy resistance exercise has been shown to be a potent stimulus for increases in muscle cell size (25,30). The data in this investigation demonstrate that heavy resistance exercise protocols elicit increases in peripheral concentrations of anabolic hormones and growth factor and it is important to recognize that the various heavy resistance exercise protocols produced different response patterns. This suggests that specific hormonal responses may be linked to specific exercise protocol characteristics. Thus, the type of heavy resistance exercise protocol utilized in training may have

important ramifications on subsequent adaptations in skeletal muscle and connective tissue.

Testosterone is a potent anabolic hormone effecting muscle tissue growth (10,22,21). It is suggested that testosterone has a direct effect on skeletal muscle and that this action is not mediated by a secondary hormone (10). Furthermore, skeletal muscle preferentially binds testosterone where receptors in the levator ani muscle and reproductive tissue have greater specificity for dihydrotestosterone (10). Each heavy resistance exercise protocol in this study increased serum testosterone concentrations above resting concentrations. However, the response patterns following the various HREPs were different. In this study it was demonstrated that all protocols do not elicit the same magnitude or frequency of serum testosterone increases even when the identical total work was performed. In Series 1, when the load was lightened from 5RM to 10RM, testosterone values were above rest only at one time point (i.e. 5 min. post-exercise). In Series 2, when the rest period was increased from 1 min. to 3 min. the magnitude and frequency of response were altered. Thus, it appears that altering single factor variables in an exercise protocol does influence the serum testosterone response patterns. Increases in testosterone consequent to an acute exercise session utilizing multiple heavy resistance exercises has been previously observed (33). Testosterone increases consequent to single exercise protocols appear to be related to the amount of muscle mass utilized in the exercise (8,12). All muscle group exercises do not result in significant increases in testosterone concentrations (12).

Differences in testosterone responses to various heavy resistance exercise protocols may influence the training adaptations which occur and our data would then help to explain variations in muscle hypertrophy resulting from

different heavy resistance training programs. Increases in testosterone which occurred during exercise and into recovery have been commonly observed in other studies following high intensity aerobic and anaerobic exercise (5,20,24,34). Typically, changes in plasma volume and reduction in clearance rates secondary to reductions in hepatic blood flow have been used to explain these acute exercise responses (3,27,34). Different from previous studies we examined a longer acute recovery time period and our data demonstrate that increases in testosterone may occur in a rebound fashion later into recovery (i.e. 90 or 120 min) following certain HREPs. It might be hypothesized that regardless of the mechanism of testosterone increase, the skeletal muscle will be exposed to elevated peripheral testosterone concentration which increases the likelihood of an interaction with potential muscle tissue receptors. Our data suggest that contributions to such mechanisms by changes in % plasma volume shifts would be primarily related to serum concentrations measured during exercise (i.e. mid and IP) where the greatest changes were observed.

It has been generally shown that heavy resistance exercise produces elevations in hGH concentrations (24,28,32). In our study, only certain heavy resistance exercise protocols produced significant elevations in hGH, which confirms suggestions by Van Helder et al. (32) that exercise variables in resistance training may play an important role in determining the response of hGH. In Series 1, when the load was reduced to 10RM, no significant elevations above rest were observed. In Series 2 (higher total work), when the load was increased (i.e. to 5RM) or the rest period length increased (i.e. to 3 min), no increases in serum hGH concentrations were observed above resting levels. It is apparent from these data that within a series, which keeps total work constant, changing one program variable alters the serum hGH response pattern.

Recent evidence has demonstrated that hyperventilation and breath holding and hypoxia stimulate significant increases in hGH (7,31,33). The stress of such maneuvers is hypothesized to mediate increases in hGH via hormonal and mechanical interoceptive mechanisms. It is possible that these types of stimulatory mechanisms are active to various degrees during heavy resistance exercise and may affect hGH responses. Our data extends the results of Van Helder et al. (32) and supports the suggestion that hypoxia or factors related to more anaerobic HREP stimulates serum hGH responses. Klimes et al. (17) had previously found little effect of changes in acid-base shifts on hGH secretion. In our study we found no consistent relationships between hGH and blood lactate. Thus, the exact nature of any anaerobic influence (i.e. as measured by blood lactate) on hGH secretion during and consequent to heavy resistance exercise remains speculative at best.

The significance of hGH on muscle growth has been established as an important influence (1,10,29). Still, it is less clear what cellular mechanisms mediate such changes in muscle cell growth. With recent biochemical characterization of the growth hormone receptor future evidence should help elucidate the molecular events involved (23). The highest elevations in hGH observed in this study were in response to the Series 2, WTP-2 protocol (high total work, 10 RM, 1 min rest). Interestingly, this protocol is similar to body building routines which are orientated to increasing muscle hypertrophy (19).

It has been demonstrated that the growth effects of hGH are likely mediated through the effects of secondary hormones [i.e. somatomedins] (2,29). Somatomedins have been hypothesized to be involved with a variety of physiological roles from muscle, bone and connective tissue growth to the aging process, and in vitro studies have shown that somatomedin-C is one of

the most potent anabolic influences on muscle cell growth (10,11). Thus, we wanted to evaluate the somatomedin-C responses to the various HREPs. To our knowledge, no other study has ever evaluated the response of SM-C to heavy resistance exercise. Neuroendocrine regulation appears to act through autocrine, paracrine and endocrine routes and has both central and peripheral regulatory roles in hGH and SM-C production (26,29).

Data from this study demonstrates that heavy resistance exercise is an effective stimulus for eliciting increased peripheral serum concentrations of SM-C. At some timepoint, every HREP employed in the present study increased serum SM-C above resting values. This was not the case for serum hGH concentrations. SM-C release has been shown to be GH dependent (28). Still, our data demonstrated that HREPs can significantly increase SM-C values above resting concentrations despite the lack of any changes in serum hGH levels. Extra-hepatic sources of somatomedins have been observed and make speculation concerning other cell sources of somatomedins plausible (16). Finally, we were unable to demonstrate any systematic relationships between serum hGH and SM-C concentrations that would lead us to believe that hGH was acting as a stimulus prior to SM-C production. However, as it has been pointed out previously by Florini et al. (11) longer term integrated (i.e. 24 hr) hGH values may be more related to SM-C values and we did not measure this time period.

In summary, this study was undertaken to provide an extensive examination of endogenous anabolic hormonal and growth factor responses which may be important in influencing changes in skeletal muscle, bone and connective tissue growth and development consequent to heavy resistance exercise. Our data show that those hormones and growth factors are responsive to heavy resistance exercise, but more importantly they are sensitive to the

manipulation of exercise variables involved with the design of specific protocols. Thus, these findings may be critical for understanding the range of adaptational changes consequent to heavy resistance training

REFERENCES

1. Allen, R.E., R.A. Merkel and R.B. Young. Cellular aspects of muscle growth: myogenic cell proliferation. J. Animal Sci. 49:115-127, 1979.
2. Berelowitz, M., M. Szabo, L.A. Froehman, S. Firestone and L. Chu. Somatomedin-C mediates growth hormone negative feedback by effects on both the hypothalamus and the pituitary. Science 212: 1279-1281, 1981
3. Cadoux-Hudson, T.A., J.D. Few and F.J. Imms. The effects of exercise on the production and clearance of testosterone in well trained young men. Eur J Appl Physiol 54: 321-325, 1985.
4. Costill, D.L. and E.L. Fox. Energetics of marathon running. Med Sci Sports 1: 81-86, 1969.
5. Cumming, D.C., L.A. Brunsting, G. Strich, A.L. Ries and R.W. Rebar. Reproductive hormone increases in response to acute exercise in men. Med. Sci. Sports Exerc. 18: 369-373, 1986.
6. Dill, D.B. and D.L. Costill. Calculation of percentage change in volume of blood, plasma and red cells in dehydration. J. Appl. Physiol. 37:247-248, 1974.
7. Djarova, R., A. Ilkov, A. Varbanova, A. Kikiforova and G. Mateev. Human growth hormone, cortisol and acid-base balance changes after hyperventilation and breath-holding. Int. J. Sports Med. 7: 311-315, 1986.
8. Fahey, T.D., R. Rolph, P. Mounsgmee, J. Nagel and S. Mortara, Serum testosterone, body composition and strength of young adults. Med. Sci. Sports. 8: 31-34, 1976.

9. Fitzgerald, P.I., J.A. Vogel, J. Milette and J.M. Foster. An improved portable hydrostatic weighting system for body composition. USARIEM Tech Report T4-88. Oct 87.
10. Florini, J.R. Hormonal control of muscle cell growth. J. Animal Sci. 61: 21-37, 1985.
11. Florini, J.R., P.N. Prinz, M.V. Vitiello and R.L. Hintz. Somatomedin-C levels in healthy young and old men: Relationship to peak and 24 hour integrated levels of growth hormone. J. Gerontology, 40: 2-7, 1985.
12. Guezennec, Y., L. Leger, F. Lhoste, M. Aymonod and P.C. Pesquies. Hormone and metabolite response to weight-lifting training sessions. Int. J. Sports Med. 7: 100-105, 1986.
13. Hakkinen, K., A. Pakarinen, M. Alen and P.V. Komi. Serum hormones during prolonged training of neuromuscular performance. Eur. J. Appl. Physiol. 53: 287-293, 1985.
14. Hakkinen, K., A. Pakarinen, M. Alen, H. Kauhanen and P.V. Komi. Neuromuscular and hormonal adaptations in athletes to strength training in two years. J. Appl. Physiol. 65: 2406-2412, 1988.
15. Hakkinen, K., A. Pakarinen, M. Alen, H. Kauhanen and P.V. Komi. Relationships between training volume, physical performance capacity and serum hormone concentrations during prolonged training in elite weight lifters. Int. J. Sports Med. 8: 61-65, 1987.
16. Han, V.K.M., A.J. D'Ercole and P.K. Lund. Cellular localization of somatomedin (insulin-like growth factor) messenger RNA in the human fetus. Science. 236: 193-197, 1987.
17. Klimes, I., M. Vigas, J. Jurcoricova and S. Nemeth. Lack of effect of acid-base alterations on growth hormone secretion in man. Endocrinologia Experimentalis, 11: 155-162, 1977.

18. Kraemer, W.J. Endocrine responses to resistance exercise. Med. Sci. Sports Exerc., 20(suppl): S152-S157, 1988.
19. Kraemer, W.J., B.J. Nobel, M.J. Clark and B.W. Culver. Physiologic responses to heavy-resistance exercise with very short rest periods. Int. J. Sports Med. 8: 247-252, 1987.
20. Kuoppasalmi, K., H. Naveri, S. Rehunen, M. Harkonen and H. Adlercreutz. Effect of strenuous anaerobic running exercise on plasma growth hormone, cortisol, luteinizing hormone, testosterone, androstenedione, estrone and estradiol. J. Ster. Biochem. 7: 823-829, 1976.
21. Kuoppasalmi, K. and H. Adlercreutz. Interaction between catabolic and anabolic steroid hormones in muscular exercise. In: Exercise Endocrinology. K. Fotherby and S.B. Pal (Eds.) Berlin: Walter de Gruyter, 1985, pp. 65-98.
22. Lamb, D.R. Androgens and exercise. Med. Sci. Sports. 7:1-5, 1975.
23. Leung, D.W., S.A. Spencer, G. Cachianes, R.G. Hammonds, C. Collins, W.J. Henzel, R. Barnard, M.J. Waters and W.I. Wood. Growth hormone receptor and serum binding protein: purification, cloning and expression. Nature, 330: 537-543, 1987.
24. Lukaszewska, J., B. Biczowa, D. Boliewicz, M. Wilk and B. Obuchowicz-Fedelus. Effect of physical exercise on plasma cortisol and growth hormone levels in young weight lifters. Endokrynologia Polska XXVII. 2: 149-158, 1976.
25. MacDougall, J.D., G.R. Ward, D.G. Sale and J.R. Sutton. Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization. J. Appl. Physiol. 43: 700-703, 1977.
26. Rosenfeld, R.G. and A.R. Hoffman. Insulin-like growth factors and their receptors in the pituitary and hypothalamus. In: Insulin, Insulin-Like

- Growth Factors, and Their Receptors in the Central Nervous System, M.K. Raizada, M.I. Phillips and D. LeRoith (Eds) New York: Plenum Press, 1987, pp. 277-295.
27. Rowell, L.B., J.R. Blackman and R.A. Bruce. Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. J. Clin. Invest. 43: 1677-1690, 1964.
 28. Skierska, E., J. Ustupska, B. Biczowa and J. Lukaszewska. Effect of physical exercise on plasma cortisol, testosterone and growth hormone levels in weight lifters. Endokrynologia Polska XXVII 2: 159-165, 1976.
 29. Sonntag, W.E., L.J. Forman, N. Miki and J. Meites. Growth hormone secretion and neuroendocrine regulation. In: Handbook for Endocrinology, G.H. Bass and H.M. Kaplan (Eds.) Boca Raton, Fl: CRC Press, Inc., 1982, pp. 35-59.
 30. Staron, R.S., R.S. Hikida, F.C. Hagerman, G.A. Dudley and R.F. Murray. Human skeletal muscle fiber type adaptability to various workloads. J. Histochem. and Cytochem. 32: 146-152, 1984.
 31. Sutton, J.R. Effect of acute hypoxia on the hormonal response to exercise. J. Appl. Physiol.: Respir. Environ. Exercise Physiol. 42: 587-592, 1977.
 32. VanHelder, W.P., M.W. Radomski and R.C. Goode. Growth hormone responses during intermittent weight lifting exercise in men. Eur. J. Appl. Physiol. 53: 31-34, 1984.
 33. VanHelder, W.P., K. Casey, R.C. Goode and W.M. Radomski. Growth hormone regulation in two types of aerobic exercise of equal oxygen uptake. Eur. J. Appl. Physiol. 55: 236-239, 1986.

34. Weiss, L.W., K.J. Cureton and F.N. Thompson. Comparison of serum testosterone and androstenedione responses to weight lifting in men and women. Eur. J. Appl. Physiol. 50: 413-419, 1983.
35. Wilkerson, J.E., S.M. Horvath and B. Gutin. Plasma testosterone during treadmill exercise. J. Appl. Physiol: Respirat. Environ. Exercise Physiol. 49: 249-253, 1980.
36. Wilmore, J.H., P.A. Vodak, R.B. Parr, R.N. Girondolf and J.E. Behing. Further simplification of a method for determination of residual lung volume. Med. Sci. Sports Exer. 12: 216-218, 1980.
37. Winter, D.A. Biomechanics of Human Movement, John Wiley & Sons, New York, 1979.

Table 1. Experimental Heavy Resistance Exercise Protocols Utilized

<u>Exercise Order</u>	<u>Repetition Maximum(RM) and Number of Sets</u>	
	<u>Series 1</u>	<u>Series 2</u>
1. Bench Press (Universal Weight Machines)	5 RM x 5 Sets	10RM x 3 Sets
2. Double Let Extensions (Universal Weight Machine)	5 RM x 5 Sets	10RM x 3 Sets
3. Military Press (Universal Weight Machine)	5 RM x 3 Sets	10RM x 3 Sets
4. Bent Leg Incline Sit-ups (Free Weights)	5 RM x 3 Sets	10RM x 3 Sets
5. Seated Rows (Universal Weight Machine)	5 RM x 3 Sets	10RM x 3 Sets
6. Lat Pull Down (Universal Weight Machine)	5 RM x 4 Sets	10RM x 3 Sets
7. Arm Curls (Free Weights)	5 RM x 3 Sets	10RM x 3 Sets
8. Leg Press (Universal Weight Machine)	5 RM x 5 Sets	10RM x 3 Sets

SERIES 1

WTP-1 = 5 RM, 3 min rest
mean total work (J) = 49,161

o Load Control 10 RM
(same rest and total work as WTP-1)

o Rest Control (1 min)
(same load and total work WTP-1)

(* = $p < 0.05$ from WTP-1)

SERIES 2

WTP-2 = 10 RM, 1 min rest
mean total work (J) = 79,859*

o Load Control (5 RM)
(same rest and total work WTP-2)

o Rest Control (3 min)
(same load and total work WTP-2)

Figure 1. Serum testosterone concentrations for the various HREP are presented. * = $p < 0.05$ from corresponding resting values.

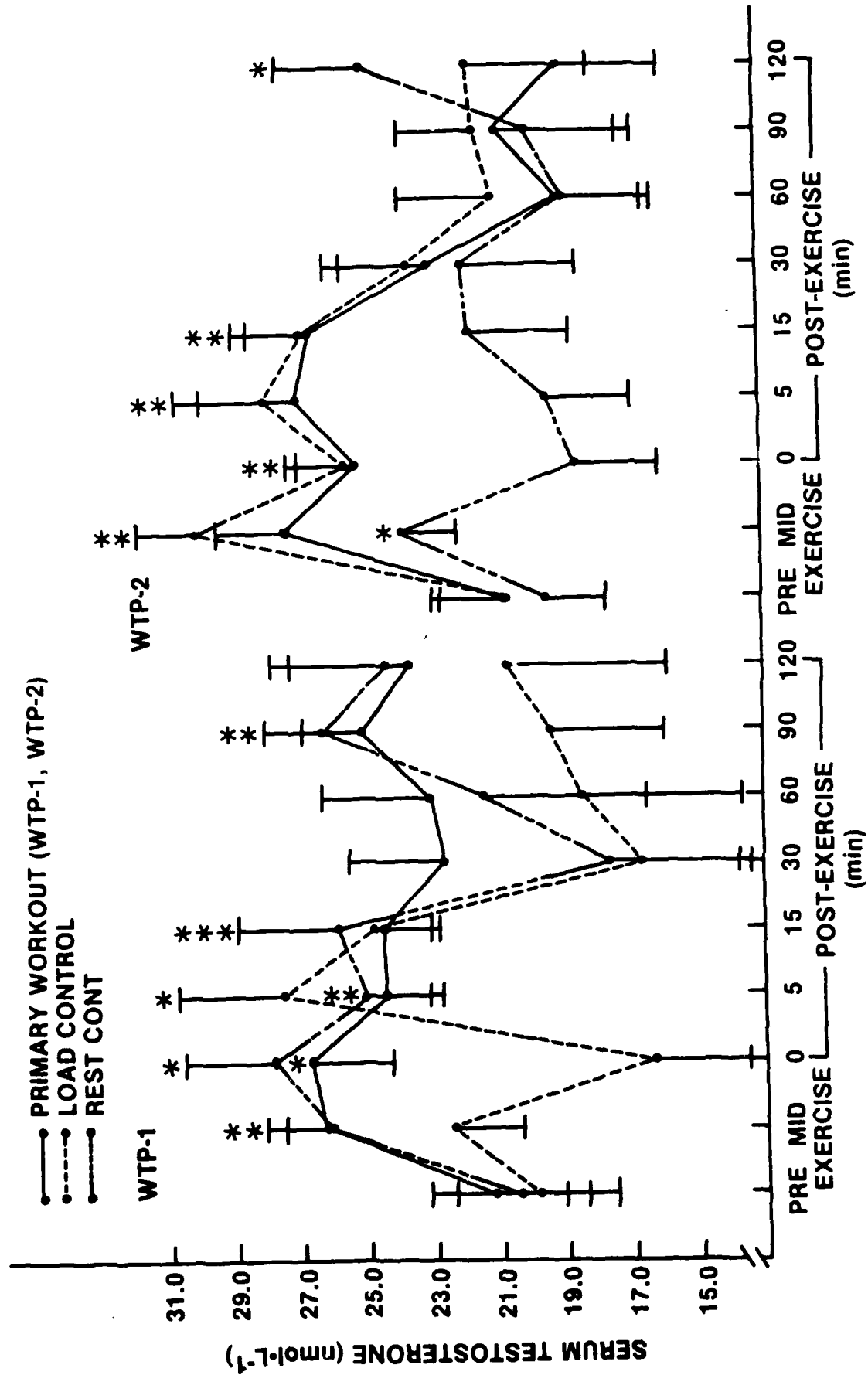


Figure 2. Serum human growth hormone concentrations for the various HREP are presented. * = $p < 0.05$ from corresponding resting values.

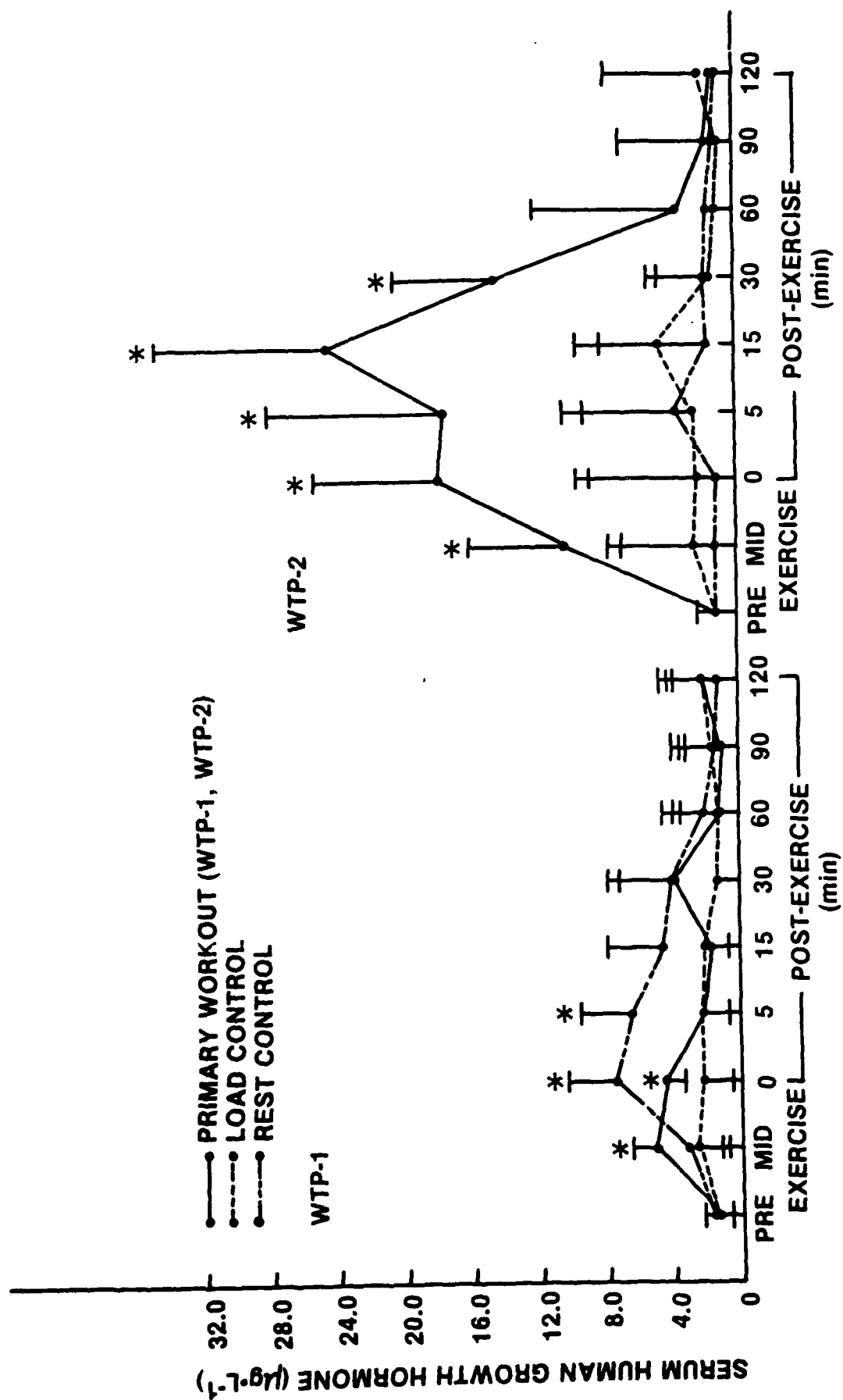


Figure 3. Serum somatomedin-C concentrations for the various HREP are presented. * = $p < 0.05$ from corresponding resting values.

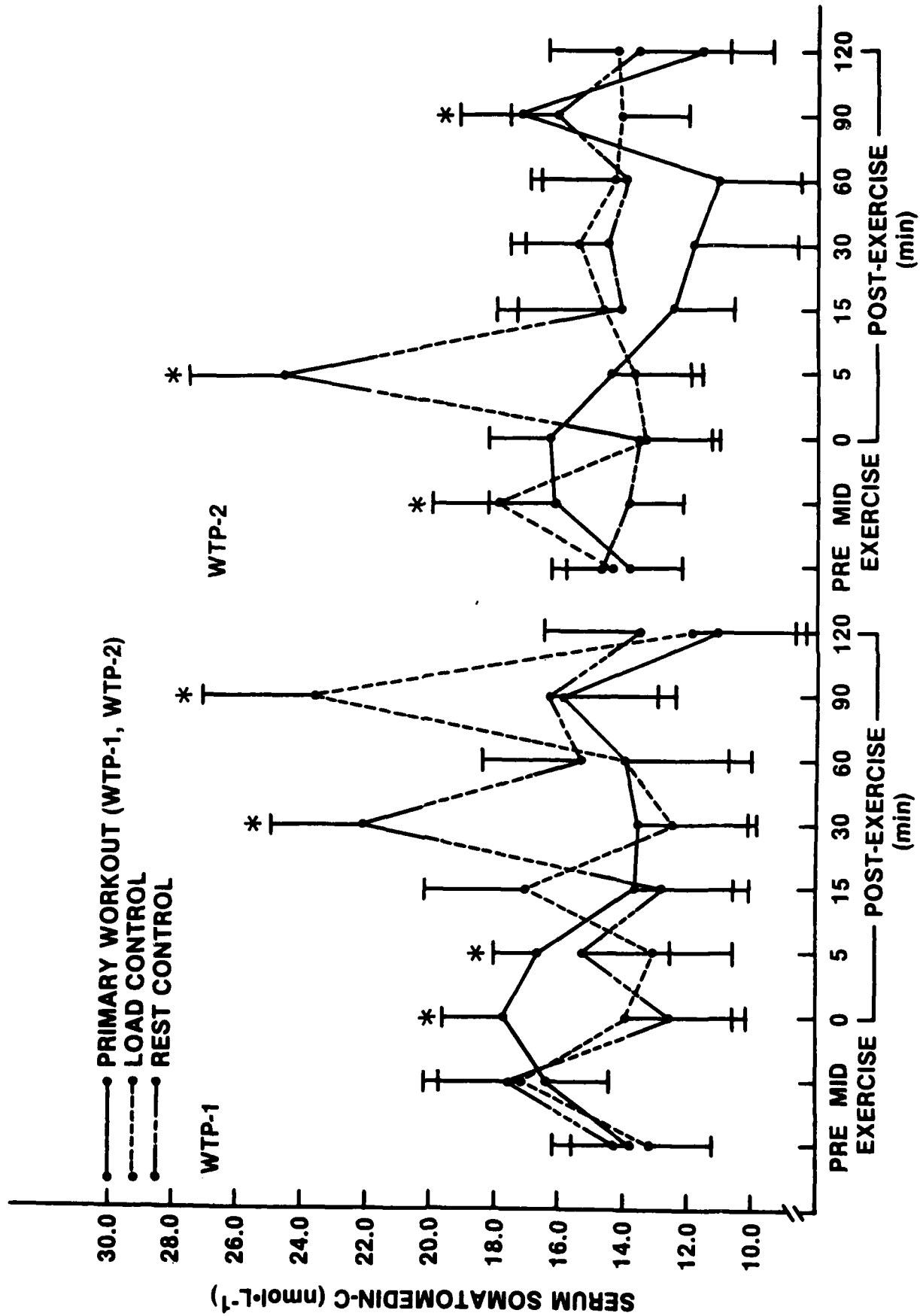


Figure 4. Serum glucose (panel A) and whole blood lactate (panel B) concentrations for the various HREP are presented. * = $p < 0.05$ from corresponding resting values.



HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.

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